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The Small Left Ventricle is an Important Predictor of Clinical Outcomes In Severe Aortic Stenosis

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Abstract: Calcific aortic stenosis (AS) is the most common valvular heart disease requiring invasive intervention. While the implantation of an artificial heart valve restores normal intracardiac hemodynamics, baseline left ventricular (LV) parameters can significantly influence both early and long-term postoperative outcomes. It is well-established that patients with LV dilation experience worse outcomes after surgical or transcatheter treatment of severe AS compared to those with normal LV dimensions. However, the impact of reduced LV volume remains insufficiently explored. This study aims to investigate the effect of small LV dimensions on clinical outcomes in the management of severe high-gradient AS, based on existing literature. We conducted a scoping review using PubMed, Google Scholar, and Elibrary databases. Articles published between 2015 and 2024 were included, though earlier publications were also referenced to support specific sections. Both original research articles and systematic reviews were examined. The central focus of this study is to investigate the impact of small LV size on clinical outcomes in patients undergoing treatment for severe high-gradient AS. According to the existing literature, small LV size is associated with significantly higher 30-day and 2-year all-cause mortality (20.8% vs. 14.3%; adjusted HR, 1.58 [95% CI, 1.20–2.09]; p = 0.0013) and cardiovascular mortality (8.8% vs. 5.5%; adjusted HR, 1.93 [95% CI, 1.25–2.98]; p = 0.0028). Furthermore, considerable emphasis has been placed on the low transvalvular flow (low flow–high gradient) pattern, which represents a critical predictor of clinical outcomes in severe AS. Notably, the 2-year cardiovascular event-free survival rate in patients exhibiting the low flow–high gradient pattern is 30 ± 12%. These findings underscore the importance of LV size and flow patterns in prognostic assessments and therapeutic decision-making for severe AS. The authors emphasize that these patterns require further investigation for early identification and minimization of perioperative risks, determination of optimal timing for surgical or transcatheter intervention, and improvement of prognosis for patients following treatment of severe aortic stenosis.

Keywords: Severe Aortic Stenosis, Aortic Stenosis Phenotypes, Small Left Ventricle, Low-Flow/High-Gradient, Diastolic Dysfunction, Valvuloarterial Impedance

1. Introduction

Calcific aortic stenosis (AS) is among the most prevalent cardiovascular diseases in developed countries, affecting approximately 3.4% of individuals over the age of 75. It is also the most common primary valvular disease necessitating invasive intervention [1,2]

The implantation of aortic valve (AV) prosthesis, by conventional or transcatheter methods, restores normal intracardiac hemodynamics. However, the baseline condition and size of the LV play a crucial role in determining postoperative outcomes. Although there is a substantial body of evidence demonstrating that patients with LV dilatation following treatment for severe AS tend to have poorer outcomes [3,4], the potential impact of low LV volume on the risk of adverse events during surgical or transcatheter interventions for severe AS has not been sufficiently studied. The optimal timing for surgical or transcatheter intervention to reduce perioperative risks and achieve the best treatment outcomes has not yet been determined. Assessing perioperative risks in patients undergoing AVR remains relevant despite the increasing prevalence of TAVR. In developing countries, open surgical intervention remains the primary treatment method. Furthermore, there are situations where TAVR is difficult or impossible to perform, such as in cases of unsuitable aortic valve anatomy or inability to access via the transfemoral route.

2. Classifications of Severe Aortic Stenosis

Patients with severe AS are traditionally categorized into subgroups based on symptoms, AV pressure gradients, and LV ejection fraction (LVEF). The clinical manifestations of AS encompass a wide spectrum of pathological and hemodynamic changes reflecting the diversity in disease presentation.

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Several well-established classification systems for AS are currently in use. The 2020 American Heart Association/American College of Cardiology (AHA/ACC) guidelines for valvular heart disease stratify AS into stages based on symptom severity and hemodynamic parameters, distinguishing between mild, moderate, and severe forms of the condition [5]. Additionally, alternative classification frameworks have been proposed [6-10], which categorize AS patients based on stroke volume index (<35 mL/m², classified as low-flow, vs. ≥35 mL/m², classified as normal-flow) and aortic gradient (<40 mmHg, classified as low-gradient, vs. ≥40 mmHg, classified as high-gradient). These criteria create four distinct patient groups: (1) Normal-flow/high-gradient (NF/HG); (2) Normal-flow/low-gradient (NF/LG); (3) Low-flow/high-gradient (LF/HG); and (4) Low-flow/low-gradient (LF/LG).

In 2017, Genereux et al. proposed a classification system for AS based on the extent of extravalvular cardiac damage, providing a framework for predicting outcomes after valve replacement [11]. The stages range from isolated AS without damage (Stage 0) to progressive involvement of the LV (Stage 1), left atrium or mitral valve (Stage 2), pulmonary vasculature or tricuspid valve (Stage 3), and finally right ventricular dysfunction (Stage 4). This classification highlights the progression of cardiac damage associated with AS and aids in guiding treatment strategies.

A modification of the above classification was proposed for patients with severe asymptomatic AS [12]. An additional criterion for Stage 1 includes a reduction in LV global longitudinal strain (GLS) to <|15%|. Elevated left ventricular filling pressure (E/e' >14) has been replaced by Stage II diastolic dysfunction, as defined by the guidelines of the American Society of Echocardiography and the European Association of Cardiovascular Imaging [13]. For Stage 4, a further criterion was added, incorporating moderate or severe reductions in stroke volume index (SVi <30 mL/m²). All other parameters and criteria remain consistent with the original AS staging classification.

In addition to the classifications discussed, numerous alternative methods for clustering patients to evaluate AS severity have been proposed by various authors [3, 14-22]. However, hemodynamic variations linked to low LV volume, often referred to as a small LV, are not adequately addressed within the frameworks of current classifications. Critical issues such as diastolic dysfunction in severe AS and pronounced LV hypertrophy remain insufficiently explored. There are no classifications or scales that account for all of the aforementioned hemodynamic variants associated with low LV volume characteristics (small LV), diastolic dysfunction, restrictive dysfunction, and significant LV hypertrophy.

Given these gaps, a focused review of the literature examining the clinical correlation between adverse outcomes in the treatment of severe AS and the concentric LV remodelling pattern is both relevant and timely.

3. Low-flow/high-gradient pattern

The low-flow/high-gradient (LF/HG) pattern is observed in approximately 3–10% of patients with severe AS, as reported by various authors [6, 8, 10, 23]. However, when employing a multiposition approach in the assessment of the AS severity (e.g., right parasternal access), the prevalence of LF/HG increases to approximately 24% [24-26]. Despite its relatively high prevalence, the impact of the LF/HG hemodynamic variant on clinical outcomes following surgical AVR or TAVR remains insufficiently studied.

In 2017, a systematic review and meta-analysis titled *Effects of Aortic Valve Replacement on Severe Aortic Stenosis and Preserved Systolic Function: A Systematic Review and Network Meta-Analysis* examined the outcomes of aortic valve replacement (AVR) in patients with severe AS. The pooled analysis of 15 studies involving 9,737 patients revealed that a low-flow pattern (both high-gradient [LF/HG] and low-gradient [LF/LG]) was associated with significantly increased mortality compared to the normal-flow pattern. Specifically, the OR for mortality was 1.88 (95% CI: 1.43–2.46) for LF/LG and 1.77 (95% CI: 1.16–2.70) for LF/HG, whereas for normal-flow subgroups, mortality was lower (NF/LG: OR 1.11; 95% CI: 0.81–1.53; NF/HG: OR 1.16; 95% CI: 0.82–1.64) [27]. Patients with low-flow AS exhibited significantly elevated levels of B-type natriuretic peptide (BNP) compared to NF/HG patients, irrespective of AV pressure gradients [10]. Notably, a study by Eleid et al. reported that valve replacement in patients with severe LF/HG AS did not result in reduced mortality following either AVR surgery or transcatheter aortic valve replacement (TAVR). Moreover, patients in the low-flow groups experienced the highest mortality risk, with 2-year survival rates without cardiovascular events being 83±6% for NF/LG, 44±6% for NF/HG, 30±12% for LF/HG, and 27±13% for LF/LG (p<0.0001) [8].

Multivariate analysis identified the LF/LG and LF/HG patterns as strong independent predictors of adverse prognosis in patients with severe AS compared with the NF/HG pattern. Specifically, LF/LG had an OR of 5.26 (95% CI: 2.04-14.3; p = 0.045) and LF/HG had an OR of 2.38 (95% CI: 1.02-5.55; p = 0.001) [10]. In addition, LF/HG AS patients had the lowest indexed valve area, along with indicators of increased vascular resistance and high-pressure gradients, highlighting a distinct pathophysiological profile within this subgroup of severe AS patients with preserved LVEF [6, 7, 9].

It appears that the LF/HG pattern, in combination with small LV dimensions due to excessive LV hypertrophy, restrictive disorders, and diastolic dysfunction, leads to poor LV filling during diastole, resulting in reduced cardiac output and stroke volume even after surgical or endovascular correction of the valve defect. This ultimately causes blood stasis in the pulmonary circulation and hemodynamic collapse.

4. Excessive LV hypertrophy as a phenotype of hypertrophic cardiomyopathy

LV wall thickness alone is an unreliable indicator of LV hypertrophy, which is instead determined by calculating LV mass indexed to body surface area (LVMI). Additionally, relative LV wall thickness (RWT) can be assessed by comparing LV wall thickness to LV end-diastolic dimension (LVEDD) [28]. Classical descriptions of LV geometric patterns consider both the LVMI and RWT. Based on these parameters, LV geometry is categorized into four types: 'normal' geometry, 'concentric remodelling' (normal LVMI with increased RWT >0.42), 'concentric LV hypertrophy' (elevated LVMI with RWT >0.42), and 'eccentric LV hypertrophy' (elevated LVMI with RWT <0.42) [29].

AS is characterized by concentric LV remodelling. When myocardial hypertrophy is inadequate to compensate for increased load, RWT rises disproportionately, leading to elevated wall stress and greater afterload, which may ultimately result in LV systolic dysfunction. Notably, preserved LVEF in the context of concentric remodelling does not accurately reflect the extent of myocardial damage. Parameters deemed optimal for an LV with normal geometry may not be applicable to one with concentric remodelling [30]. Even in the presence of preserved LVEF, patients with severe AS, whether symptomatic or asymptomatic, face a significant risk of mortality and should be evaluated for AVR [31, 32].

LV geometry and function vary according to loading conditions. On the one hand, LV may have a small cavity and hypertrophied walls, which is predominantly observed in women, as they are characterised by normal or even supernormal shortening fraction in combination with low LV wall stress [33, 34]. In men, LV is more often dilated due to relatively thin walls and lower shortening fraction due to high wall stress [35]. In addition, LV wall tends to be thicker in older age groups ($p < 0.001$) [36]. Eccentric LV hypertrophy is more prevalent in the LF/LG (28.6%) and NF/LG (30.4%) groups compared to NF/HG (8.9%) and LF/HG (16.7%) groups, while the proportion of patients with concentric LV hypertrophy does not differ significantly among these groups [37].

LV hypertrophy develops as an adaptive response to increased systolic pressure. Severe LV hypertrophy is defined as a myocardial mass exceeding the expected value by more than 110%, adjusted for the patient's height, sex, and wall stress [38]. This form of inadequate LV hypertrophy is an independent predictor of high perioperative mortality. In the study *Prognostic Effect of Inappropriately High Left Ventricular Mass in Asymptomatic Severe Aortic Stenosis*, patients with inappropriately high LV mass (iLVM) reached key endpoints (including all-cause mortality, AVR, or hospitalization for non-fatal myocardial infarction and/or congestive heart failure) at twice the rate of patients with appropriate LV mass (aLVM) – 67% versus 30% ($p < 0.001$). The survival rates for patients with aLVM and iLVM were 78% vs. 56% at 1 year, 68% vs. 29% at 3 years, and 56% vs. 10% at 5 years (all $p < 0.01$) [39].

Patients with AS not only develop LV myocardial hypertrophy but also experience increased connective tissue volume. The accumulation of myocardial collagen, along with the enhanced expression of genes responsible for the synthesis of collagen I and III and fibronectin, is closely linked to the activation of the renin-angiotensin-aldosterone system (RAAS). Both collagen and fibronectin gene expression are directly correlated with LV end-diastolic pressure and inversely correlated with contractile function. Following AV surgery, the reduction in RAAS activity plays a key role in reversing LV remodelling and promoting regression of hypertrophy. This process leads to normalization of LV function, with a notable reduction in LV myocardial mass by 20-30% observed within the first 6-12 months post-surgery [2].

Excessive LV hypertrophy can lead to a significant reduction in LV volume and the development of a phenotype resembling hypertrophic cardiomyopathy (HCM), which in turn increases perioperative risks and worsens long-term prognosis. Distinguishing between myocardial hypertrophy caused by AS and that resulting from a combination of AS and HCM can be challenging. Patients with AS exhibiting the HCM phenotype have a notably higher incidence of cardiovascular events, as well as increased in-hospital and long-term mortality, compared to patients without the phenotype following TAVR. These events include a more than seven-fold increase in aortic dissection and a greater than four-fold increase in cardiogenic shock and vascular complications. Additionally, LV outflow tract obstruction in severe AS is associated with an unfavorable prognosis and may necessitate interventions such as alcohol septal ablation before TAVR or surgical excision of septal hypertrophy during AVR surgery [40-44].

After AVR, concentric hypertrophy and small LV dimensions create a hyperdynamic state. A sudden decrease in afterload increases LV contractility, enhancing systolic movement of the anterior mitral valve leaflet (SAM syndrome) and dynamic LV outflow tract obstruction. This leads to the development of mitral regurgitation, hypotension, and acute hemodynamic collapse. Additionally, the hypertrophied, fibrotic myocardium in small ventricles increases the risk of ventricular arrhythmias. Case reports have documented ventricular fibrillation following TAVR, often triggered by ischemia or acute obstruction [45, 46].

5. Small LV

LV size is a critical prognostic marker in various cardiovascular diseases. Statistically, the reference range for LV size is derived from the 95th percentile of the normal population, excluding the extreme 5% at both upper and lower limits [47]. LV dimensions demonstrate a relationship with survival rates; as LV size increases, cardiovascular outcomes progressively worsen. Specific threshold values are established primarily for clinical and practical

applications [28]. While much attention has been given to LV cavity enlargement – acknowledged as a predictor of poor outcomes in cardiogenic pathology [3, 4] – the clinical implications of a small LV remain inadequately explored.

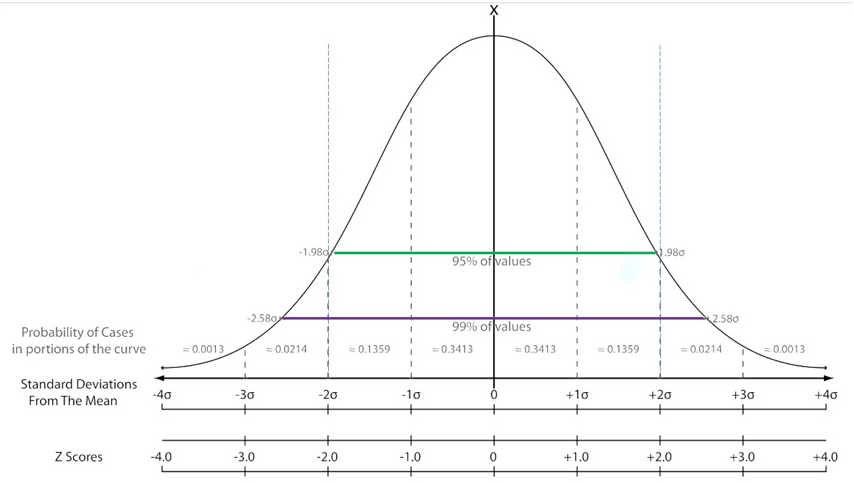

Figure 1: Normal distribution curve. The reference limits can be calculated using the mean value of a parameter in the population and the standard deviation. The range covering two standard deviations above and below the mean includes 96.4% of all "normal" subjects. Similarly, three standard deviations cover 99.7% of the normal population [28].

A small LV is currently defined as an LVEDD of <42.0 mm for men and <37.8 mm for women, according to the American Society of Echocardiography recommendations [48]. However, it is important to note that individuals of East Asian or Indian descent typically have slightly smaller LV volumes, which should be considered in clinical practice [28]. A 2021 study assessed the impact of a small LV on clinical outcomes in 2,584 patients undergoing TAVR. Among the 466 patients with a small LV, 30-day mortality was significantly higher (18.0%), and this group also showed higher 2-year all-cause mortality (20.8% vs. 14.3%; adjusted HR, 1.58 [95% CI, 1.20-2.09]; p = 0.0013) and cardiovascular mortality (8.8% vs. 5.5%; adjusted HR, 1.93 [95% CI, 1.25-2.98]; p = 0.0028). Furthermore, small LV dimensions were associated with poorer clinical outcomes after TAVR, independent of low flow and LV hypertrophy. Patients with a small LV also experience higher mortality and perioperative complication rates following AV surgery, with myocardial hypertrophy being a key contributor to the development of diastolic dysfunction [48].

In AS, small LV dimensions are frequently associated with low stroke volume (SV) and reduced cardiac output (CO). Following aortic valve replacement, the inability to augment cardiac output due to fixed LV dimensions can exacerbate heart failure and elevate mortality risk [48]. Additionally, the degree of afterload prior to defect correction is inversely correlated with the ability to maintain stable hemodynamics post-AVR. Factors such as increased LV stiffness, myocardial fibrosis, and impaired diastolic filling contribute to the rapid deterioration of hemodynamic function.

6. Diastolic dysfunction worsens prognosis in patients with severe AS

Diastolic dysfunction (DD) manifests earlier than LVEF reduction in patients with AV disease and serves as both a marker of AS severity and decompensation, as well as a key determinant of clinical outcomes following surgical or endovascular AV interventions [49-53]. Impaired relaxation occurs in both concentric and eccentric LV hypertrophy, but chamber stiffness is specifically increased in concentric LV hypertrophy [49]. The progression of DD from grade 1 to grade 3 is associated with marked deterioration in myocardial structure and function. Recent studies reveal that grade 2 or higher DD affects approximately 42% of patients with severe AS and is strongly correlated with increased cardiovascular mortality and hospitalization rates [51]. Furthermore, left atrial strain, an additional marker of LV diastolic function, is independently associated with elevated rates of hospitalization and mortality in patients with moderate to severe AS [2, 54, 55].

In grade 3 DD, characterized by the restrictive pattern, LV pressure becomes so elevated that LV filling occurs predominantly during the early diastolic phase, with negligible filling in subsequent phases. This results in a rapid rise in LV pressure, cessation of transmitral blood flow, and a peak E wave velocity exceeding the peak A wave velocity by more than 1.5 times [56]. The restrictive DD pattern is an independent predictor of cardiovascular mortality and re-hospitalization within one year (OR: 2.24; 95% CI: 1.34-3.76), though its prognostic value diminishes beyond two years (OR: 1.48; 95% CI: 0.85-2.58) [51].

Following AVR, LV hypertrophy undergoes significant regression, primarily driven by a reduction in myocardial muscle tissue, while the total amount of fibrotic tissue within the LV remains unchanged [57–60]. Small LVs have a limited capacity for reverse remodeling after AVR. Concentric myocardial hypertrophy and fibrosis restrict diastolic filling, maintaining elevated LV pressure and resulting in pulmonary circulation congestion. The

relative increase in fibrous tissue may transiently worsen LV diastolic dysfunction shortly after valvular obstruction resolves [61]. Although earlier studies suggested the possibility of long-term normalization of LV diastolic function [62], more recent evidence challenges these findings [59].

In addition to conventional echocardiographic parameters for evaluating AV stenosis, advancements in diagnostic techniques are enhancing the early detection of severe AS, enabling timely surgical or transcatheter treatment [63, 64].

7. Current Diagnostic Approaches for Severe AS

Clinical decision-making and the determination of surgical indications for patients with severe AS are traditionally based on LVEF [1, 5]. However, LVEF can remain within normal limits despite the presence of concentric remodelling, LV myocardial hypertrophy, or the development of chronic heart failure with preserved LVEF, limiting its sensitivity in detecting severe AS. Recent guidelines from the National Institute for Health and Care Excellence (NICE) in the UK recommend adopting an LVEF threshold of <55%, which enhances the sensitivity of this parameter for identifying subclinical LV systolic dysfunction [2].

Various diagnostic approaches have been proposed for the early detection of LV dysfunction in AS. Speckle-tracking echocardiography enables the estimation of GLS, with values <|15%| indicating higher risk of adverse outcomes [65, 66, 67]. The assessment of the first phase of LV ejection fraction (EF1), which represents the change in LV volume from end-diastole to peak flow at the AV, is another valuable parameter; a value <25% is linked to an increased risk of complications [68].

The Energy Loss Index (ELI) is a parameter that adjusts the aortic valve area (AVA) for pressure recovery, calculated using the formula [(AVA × AA) / (AA - AVA)] / BSA, where AA is the cross-sectional area of the aorta at the sinotubular junction, and BSA is the body surface area [69, 70]. From a physiological perspective, ELI offers an advantage over Doppler-measured AVA or pressure gradient, as it better reflects the actual energy loss caused by AS, thereby providing a more accurate indication of the increased ventricular workload [71]. Currently, ELI has demonstrated significant prognostic value in asymptomatic AS patients [72].

Calculation of AVA using the continuous flow equation may underestimate the actual valve area due to the elliptical shape of the LV outflow tract [73]. Furthermore, the angulation of the aortic root can hinder the proper alignment of the ultrasound beam from the apical position, potentially causing inaccuracies in the evaluation of velocity indices across the AV. Patients with acute aortic root angulation are more likely to exhibit a higher peak velocity (Vmax) in the right parasternal window (65% vs. 43%; $p = 0.05$) and less likely to show higher Vmax in the apical window (19% vs. 48%; $p = 0.005$) [74].

Valvuloarterial impedance (Zva) is a diagnostic parameter used to assess total left ventricular (LV) afterload and predict subsequent LV dysfunction [75]. While similar to AV impedance, Zva incorporates LV afterload caused by systemic vascular resistance. Under conditions of relatively normal arterial pressure and systemic vascular resistance, Zva demonstrates a strong correlation with the severity of AS, even in the presence of transvalvular flow variability. This parameter is particularly useful in identifying cases of paradoxical LF/LG AS, though its broader clinical implications remain a subject of debate.

A Zva value ≥5.0 mmHg·ml·m² may indicate LV afterload mismatch and LV systolic dysfunction, while a Zva ≥5.5 mmHg·ml·m² is associated with a 2.5-fold increase in overall mortality. This parameter is particularly valuable in patients who do not meet the criteria for severe aortic stenosis AS, such as those with severe LF/LG AS and preserved LVEF. Additionally, a Zva >3.5 mmHg·ml·m² serves as a predictor of adverse outcomes in asymptomatic patients with severe AS, reflecting the presence of LV systolic dysfunction and DD. Thus, Zva plays a crucial role in risk stratification and informs management strategies for patients with AS who fall outside the standard diagnostic thresholds for severe disease [76-80].

An important aspect of diagnosing severe AS is the application of a multi-position approach during echocardiography. This approach involves using different echocardiographic views, such as right parasternal or subcostal, to assess the hemodynamic profile of patients. For example, patients initially categorized in the LF/LG group may shift to the LF/HG group when these additional views are incorporated [24-26]. Despite its potential to provide more accurate assessments, the multi-position approach remains underutilized in clinical practice. As a result, in patients with small LV, the transvalvular gradients may be underestimated prior to surgical AVR, leading to challenges in proper risk stratification and decision-making for intervention.

In many patients, echocardiography alone provides sufficient information for assessment, however, additional imaging may be advantageous in certain cases. In patients with discordant findings of severe AS on echocardiography, further imaging such as computed tomography (CT) or stress echocardiography in low-flow patients can help clarify AS severity and guide treatment decisions. For those with suspected aortopathy, CT or magnetic resonance imaging (MRI) is recommended for a comprehensive evaluation of the thoracic aorta. If amyloidosis is suspected, MRI or bone scintigraphy should be considered. Additionally, CT angiography is routinely performed before TAVR to assess patient suitability for the procedure and determine the most appropriate access route [2].

In recent years, there has been increasing interest in using myocardial fibrosis assessment as an early, objective marker of LV decompensation, particularly in asymptomatic patients. Advances in imaging techniques have

enabled the reliable noninvasive detection of myocardial fibrosis [81, 82]. Several studies have shown that replacement fibrosis, which is irreversible, can be detected by late gadolinium enhancement (LGE) on cardiac MRI, and that this finding correlates strongly with histological results from myocardial biopsy [83, 84]. Diffuse fibrosis, which occurs earlier and is potentially reversible, can be quantified using T1 mapping techniques on MRI [85]. A prospective cohort study conducted in 2017 quantified total myocardial extracellular volume indexed to body surface area (iECV) from cardiac MRI data, revealing that the upper limit of normal iECV in a control group was 22.5 mL/m². The iECV index showed a strong correlation with diffuse histological fibrosis ($r = 0.87$; $p < 0.001$) and was significantly elevated in patients with AS (23.6 ± 7.2 mL/m² vs. 16.1 ± 3.2 mL/m² in controls; $p < 0.001$) [86].

A 10-year prospective study evaluating patients who underwent AVR for symptomatic severe AS assessed the impact of myocardial replacement fibrosis (MRF) on long-term outcomes. All-cause mortality was observed in 38.9% (n=21) of the cohort, with rates of 14.3% (n=3) in the group without MRF, 42.9% (n=6) in those with mild MRF, and 63.2% (n=12) in those with severe MRF (p=0.006). Survival was lowest among patients with severe MRF, as indicated by a log-rank P-value of 0.003. MRF emerged as an independent predictor of adverse outcomes (OR, 1.271; 95% CI, 1.032–1.564; p=0.024). After 10 years of follow-up, no regression of replacement fibrosis was observed in any patient within the entire cohort [87].

These findings highlight the need to identify new, objective markers of early LV decompensation to improve the timing of surgical interventions and enable effective monitoring of myocardial status over time.

The diagnosis of severe AS is typically based on standard echocardiographic examination. The use of an extended echocardiographic protocol, in line with current guidelines, in routine clinical practice could enhance diagnostic capabilities. This would allow for a more accurate determination of perioperative risks and better optimization of the timing for surgical AV intervention in patients with severe AS.

In patients with extreme LV hypertrophy, diastolic dysfunction, and restrictive abnormalities, it may be beneficial to include mandatory valvuloarterial impedance assessment in the echocardiography protocol for a more precise evaluation of the "threshold" afterload. It appears that the higher the afterload and the greater the LV restriction, the more likely the patient is to be classified as high surgical risk. A staged approach to intervention may be considered, starting with balloon dilation of the aortic valve to reduce afterload to some extent, followed by AVR or TAVR. In cases of extreme hypertrophy with an HCM pattern, multidisciplinary strategies, including preventive septal reduction or staged procedures, are critically important.

It is important to emphasize that no single method is ideal for evaluating patients with aortic stenosis. Therefore, it is crucial to employ a combination of all available diagnostic techniques.

8. Conclusion

AS remains one of the most prevalent cardiovascular conditions in developed countries, with various classification systems proposed to stratify patients with severe AS. However, current classifications do not fully address the hemodynamic variants of severe AS associated with low LV volume, such as the small LV phenotype. There is limited data on the relationship between this phenotype, diastolic dysfunction, and critical LV hypertrophy. Therefore, further investigation into the clinical implications of small LV dimensions and their impact on patient outcomes following surgical intervention is warranted.

Evidence suggests that the LF/HG pattern is a strong independent predictor of poor prognosis, underscoring the need for more research to assess perioperative risks, optimize surgical timing, and enhance long-term outcomes for patients with severe AS undergoing AVR.

Patients with small LV size likely require a more careful assessment of surgical risk. Small LV has a limited capacity for reverse remodeling. After AVR, the inability to increase cardiac output due to fixed LV size may lead to acute hemodynamic collapse, exacerbate heart failure, and increase mortality.

The authors intend to further investigate this topic and develop a prognostic model for patients with severe AS. Additionally, they aim to provide specific recommendations and monitoring strategies for clinicians and propose various protocols for invasive interventions, particularly in the management of patients with small LV dimensions and diastolic dysfunction. The clinical implications, especially in terms of managing patients with small LV sizes, will be explored in more detail, and the potential for applying the findings to other patient populations will also be discussed.

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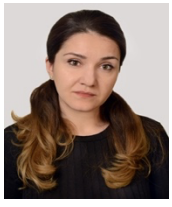

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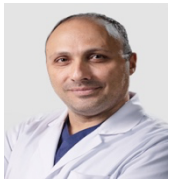

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